H. somnus could be reisolated for several weeks after infection. The mild, persistent mucopurulent vaginitis reproduced in this study was similar to that reported in field outbreaks of disease in Australia (Webber *et al* 1983; Patterson *et al* 1984) and strongly supports results of similar experimental infections by Klavano (1980) and Miller and Barnum (1983).

Histopathological lesions in the reproductive tract suggest an inflammatory and immunological response to foreign antigen(s) or pathogen(s), but a pathogenic relationship between lesions and *H. somnus* cannot be confidently established as there was a long time interval between infection and necropsy. Similar vulval lesions were reported by Crawley *et al* (1950) who attributed the cause to a "*Haemophilus*-like" pleomorphic rod. Vestibular adenitis and squamous metaplasia in all 5 glands examined, with isolation of *H. somnus* from the vestibular gland of one heifer, killed soon after infection, may support suggestions of Miller *et al* (1983a) that the vestibular gland could be a reservoir of *H. somnus* infection. In general, the more severe gross and histopathologic lesions observed in the earlier killed heifers (C, G), corresponded with increased isolation of *H. somnus*.

Systemic immunisation 3 weeks prior to experimental infection resulted in a rise in humoral antibody levels in 2 of 3 heifers, but antibody was not demonstrated in vaginal mucus at the time of infection. Antibody did however appear in vaginal mucus at oestrus 3 weeks after infection coinciding with vaginal inflammation and discharge. Conditions during the oestrous cycle thus appear to be important in the expression of disease. Immunisation may therefore have the potential to prevent disease if given more than one oestrous cycle before infection.

The results indicate that a potentially pathogenic strain of *H. somnus* can reside in the vagina in the apparent absence of clinical disease. Host factors may therefore need to be closely considered in studying the pathogenicity and immunity of *H. somnus* infection. The role of the bull as a reservoir of infection, as suggested by Corboz (1981) and Humphrey and Stephens (1983) and the occurrence of venereal transmission, together with initial low levels of local immunity at oestrus, may explain the apparent high prevalence of disease in infected herds after mating. Further studies on pathogenesis, host response and immunity are necessary.

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References

Crawley, J F, Wills, C G and MacGregor, K L (1950)—*Can J comp Med* 14: 5

Corboz, L (1981)—*Haemophilus, Pasteurella and Actinobacillus* Academic Press, London, p 133

Humphrey, J D and Stephens, L R (1983)—*Vet Bull, Weybridge* 53: 987

Klavano, G G (1980)—*Proc Soc Theriogenol, Nebraska*, p 139

Miller, R B and Barnum, D A (1983)—*Vet Path* 20: 584

Miller, R B, Barnum, D A and McEntee, K E 1983a)—*Vet Path* 20: 515

Miller, R B, Lein, D H, McEntee, K E, Hall, C E and Shin, S (1983b)—*J Am Vet Med Ass* 182: 1390

Patterson, R M, Hill, J F, Shiel, M J and Humphrey, J D (1984)—*Aust Vet J* 61: 301

Stephens, L R, Humphrey, J D, Little, P B and Barnum, D A (1983)—*J Clin Microbiol* 17: 728

Stephens, L R, Little, P B, Wilkie, B N and Barnum, D A (1984)—*Am J Vet Res* 45: 234

Webber, J J, Pritchard, D H, Gordon, L M and Dunn, H F (1983)—*Aust Vet J* 60: 276

Webber, J J and Pritchard, D H (1984)—*Haemophilus somnus/ Histophilus ovis*, Proc, Standing Committee on Agriculture Workshop, Hamilton, Victoria

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Time required for elevated blood lead concentrations to return to normal in dogs

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Estimation of lead concentration in blood is thought to be the most definitive test available for the antemortem diagnosis of exposure to lead in dogs (Zook 1978). In the absence of lead chelation therapy and after cessation of exposure to lead, the blood lead concentration can take months to years to return to normal (Maxfield *et al* 1975).

This paper records the time required for blood lead concentrations to return to within the normal (pre-treatment) values in dogs after single or repeated oral doses of a mixture of lead salts. It also records lead concentrations in liver, kidney and bones of the lead-exposed dogs in which the blood lead concentrations were allowed to return to the pre-treatment levels without the aid of chelation therapy.

Four 8-month-old male dogs were used for this experiment. They were treated with anthelmintics (praziquantel and pryantel pamoate) and were vaccinated against distemper and canine hepatitis. The mixture of lead salts was that used by Hamir *et al* (1981).

Dog A was given one oral dose of 30 mg/kg bodyweight of the lead salt mixture and dog B was given one oral dose of 60 mg/kg bodyweight.

Dogs C and D received the mixture of lead salts at an initial oral dose rate of 5 mg/kg/day for 5 weeks. The lead dose was then increased to 15 mg/kg/day for the next 6 weeks followed by a period of 4 weeks when no lead was given. For a final 2 weeks, the mixture of lead salts was administered at a dose rate of 30 mg/kg/day (Hamir *et al* 1983). The dogs were weighed weekly and the lead dose was calculated on this weight.

Before commencing the experiment, blood samples were obtained from all dogs to determine the pre-treatment blood lead concentration. The dogs were then bled on weekly basis after the cessation of lead exposure, that is, the start of experiment, until the blood lead values had reduced to the pre-treatment levels. At the end of the experimental period (9 months) dogs C and D were killed and samples of liver, kidney, distal radius and calvarium were obtained for determination of lead content. The method of blood and tissue lead analysis has been described elsewhere (Hamir *et al* 1981).

Throughout the experiment, all dogs remained healthy and none showed any clinical signs of lead intoxication. The blood lead concentrations are shown in Figure 1 (acute exposure) and in Figure 2 (chronic exposure).

In all dogs the blood lead concentration decreased markedly in the first few weeks of the experiment. The decrease thereafter was not only gradual but was quite variable from week to week. In dogs A and B the blood lead concentration decreased from 24 and 41 μg/dl to 17 and 23 μg/dl respectively at the end of the fourth week. At the end of 11 weeks these declined to 7 and 11 μg/dl in dog A and B respectively (Figure 1).

In dogs C and D the pattern of decrease in blood lead concentration was similar but required 9 months to reach the pre-treatment concentrations of 19 and 20 μg/dl (Figure 2).

Tissue lead concentrations of dogs C and D at the end of 9 months are shown in Figure 2. The lead concentrations in

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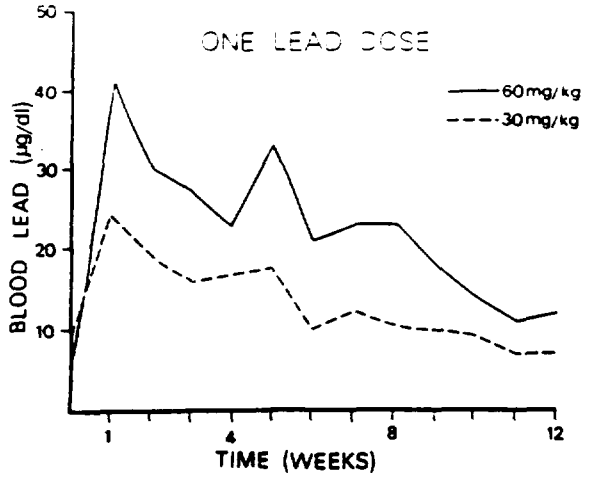

Figure 1. Blood lead concentrations of dog A (given one oral dose of 30 mg/kg lead) and dog B (one oral dose of 60 mg/kg) during the 12 weeks after dosing.

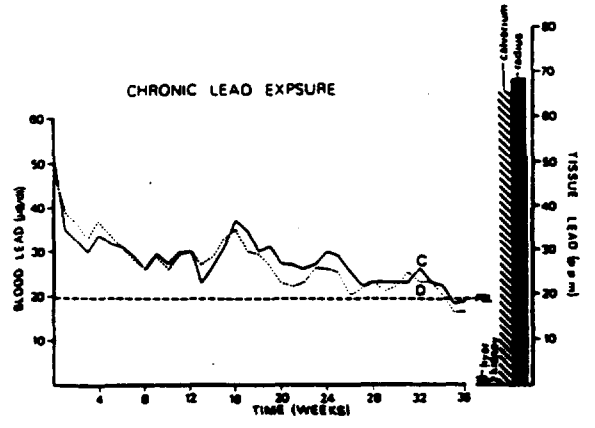

Figure 2. Blood lead concentrations of dogs C and D (given lead for prolonged period) during 9 months then following cessation of lead administration. Also illustrated are the mean tissue lead levels of these dogs at the end of the experiment. (PBL = pre-treatment blood level values).

liver and kidney were within the normal range, but the levels in the bones (radius and calvarium) were considerably higher than has been observed in dogs not exposed to lead in the Werribee area (Hamir *et al* 1986).

Lead can be absorbed from the respiratory and gastrointestinal tracts (Oehme 1972). However, only 6 to 10% of ingested lead is actually absorbed from the alimentary tract, and an even smaller proportion of this is retained in the body (Kehoe 1961). Approximately 40% of the lead retained in the body is deposited in soft tissues and 60% is located in bones (Oehme 1972) where it is relatively strongly bound. The present study shows that after administration of an oral dose of lead several months are required for the blood lead concentration to return to normal (pre-treatment) values. The study also shows that even after the blood lead concentration has reverted to the original pre-treatment value, there still remains large amounts of lead in bones, which would require several more years to eliminate from this tissue (Goodman and Gilman 1975; Freeman 1970).

References

Freeman, R (1970)—*Med J Aust* 1: 648

Goodman, L S and Gilman A (1975)—*Pharmacological Basis of Therapeutics*, 5th edn, Macmillan, New York, p 938

Hamir, A N, Handson, P D, Sullivan, N D and Anderson, D (1986)—*Vet Rec* 118: [illegible]

Hamir, A N, Sullivan, N D, Handson, P D, Wilkinson, J S and Lavelle, R B (1981)—*Aust Vet J* 57: 401

Hamir, A N, Sullivan, N D, Wilkinson, J S and Handson, P D (1983)—*Aust Vet J* 60: 372

Kehoe, R A (1961)—*J Royal Inst Pub Hlth and Hyg* 24: 81

Maxfield, M E, Stopps, G J, Barnes, J R, Snee, R D, Finan, M and Azar, A (1975)—*Am Ind Hyg Ass J* 36: 193

Oehme, F W (1972)—*Clin Toxi* 5: 151

Zook, B C (1978)—*Toxicity of Heavy Metals in the Environment*, edited by F W Oehme, Marcel Dekker, New York, p 179

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Mycoplasmosis in racing pigeons

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Racing pigeons *(Columba livia)* must have considerable endurance for long distance flying and respiratory distress will interfere with performance. This article reports our observations on 20 racing pigeons affected with respiratory disease associated with *Mycoplasma* sp.

The 20 racing pigeons came from 12 flocks. The size of the flocks varied from 12 breeding pairs, 10 yearlings and 40 youngsters, to 40 breeding pairs and 100 yearlings. The clinical signs in these flocks included loss of weight of adults, poor performance in races, and severe respiratory distress after exercise. During a series of races one owner reported that 80 out of 100 pigeons had failed to return home, and those that did arrive were severely distressed and had performed poorly.

Clinical examination confirmed that the pigeons had respiratory distress and some had conjunctivitis. Necropsy examination revealed that all affected pigeons had excess mucus in the tracheas and thickened air sacs. Histological examination of the tracheas showed that there was lymphoid hyperplasia in the lamina propria, with hyperplasia and metaplasia of the mucosal epithelium, and a reduced number of goblet cells. None of the affected air sacs was examined histologically. Using standard bacteriological techniques a variety of bacteria were isolated from some affected air sacs, including *Escherichia coli, Pasteurella gallinarum, Staphylococcus epidermidis* and *enterococci.*

Swabs from affected tracheas, air sacs and/or conjunctivae were cultured on sheep blood, McConkey and mycoplasma agar and in mycoplasma broth (Shiel *et al* 1982). All plates were incubated at 37°C, the mycoplasma agar being placed in an atmosphere of 5% CO_2, and were observed daily for growth. Colonies with typical mycoplasma morphology on agar plates were tentatively identified by indirect immunofluorescence (Del Guidice *et al* 1967). The identification of the organism was confirmed by growth inhibition tests (Clyde 1964).

These pigeons did not have chlamydiosis, salmonellosis, trichomoniasis, coccidiosis, nor external or internal parasitism. *Pasteurella multocida* was isolated from the trachea of one pigeon. Two affected pigeons also had lymphosarcomas.

These 20 pigeons yielded *Mycoplasma columbinum* (15 isolates), *M. columborale* (13), *M. synoviae* (3) and *M. gallinarum* (1). Isolations from more than one site were made from 7 pigeons; and 10 pigeons had mixed infections due to either *M. columbinum* and *M. columborale* (9 pigeons), or *M. columbinum* and *M. synoviae* (1). *M. colubinum, M. columborale* and *M. synoviae* were isolated from conjunctivae (1 isolate of each), tracheas (8, 6 and 1 respectively), and air sacs (6, 2, 1). The *M. gallinarum* was isolated from a trachea.